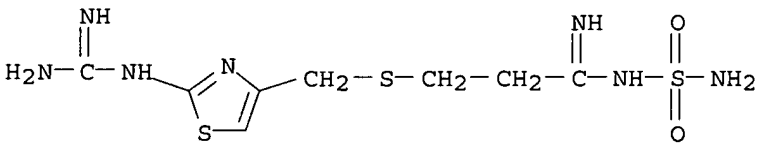
L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 76824-35-6 REGISTRY
CN Propanimidamide, 3-[[[2-[(aminoiminomethyl)amino]-4-thiazolyl]methyl]thio]-N-(aminosulfonyl)- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 3-[(2-Diaminomethyleneaminothiazol-4-yl)methylthio]-N-sulfamoylpropionamidine
CN Amfamox
CN Dispromil
CN Famodil
CN Famodine
CN Famosan
CN **Famotidine**
CN Famoxal
CN Fanosin
CN Fibonel
CN Ganor
CN Gaster
CN Gastridin
CN Gastropen
CN Ifada
CN Lecedil
CN MK 208
CN Motiax
CN Muclox
CN N-(Aminosulfonyl)-3-[[[2-[(diaminomethylene)amino]-4-thiazolyl]methyl]thio]propanimidamide
CN Nulcerin
CN Pepcid
CN Pepcid AC
CN Pepcid PM
CN Pepcidina
CN Pepcidine
CN Pepdine
CN Pepdul
CN Peptan
CN Ulcetrax
CN Ulfamid
CN Ulfinol
CN YM 11170
FS 3D CONCORD
MF C8 H15 N7 O2 S3
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DIOGENES, DRUGPAT, DRUGU, EMBASE, HSDB*, IFICDB, IFIUDB, IPA, MEDLINE, MRCK*, MSDS-OHS, NIOSHTIC, PHAR, PHARMASEARCH, PROMT, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



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PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

1129 REFERENCES IN FILE CA (1957 TO DATE)
37 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
1132 REFERENC

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 73590-58-6 REGISTRY
CN 1H-Benzimidazole, 5-methoxy-2-[[(4-methoxy-3,5-dimethyl-2-pyridinyl)methyl]sulfinyl]- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN (.+-.)-Omeprazole
CN 2-[[(3,5-Dimethyl-4-methoxy-2-pyridyl)methyl]sulfinyl]-5-methoxy-1H-benzimidazole
CN Acidex
CN Antra
CN Antra MUPS
CN Audazol
CN Aulcer
CN Belmazol
CN Ceprandal
CN Desec
CN Dizprazol
CN Dudencer
CN Elgam
CN Emeproton
CN Epirazole
CN Gastrimut
CN Gastroloc
CN Gastrozole
CN Gibancer
CN H 168/68
CN Indurgan
CN Inhibitron
CN Inhipump
CN Logastric
CN Lomac
CN Losec
CN Mepral
CN Miol
CN Miracid
CN Mopral
CN Ocid
CN Omapren
CN Omebeta 20
CN Omed
CN Omedar
CN OMEP
CN Omepradex
CN Omepral
CN Omeprazen
CN **Omeprazole**
CN Omeprazon
CN Omepril
CN Omezol
CN Omezzol
CN Omid
CN Omisec
CN Omizac
CN OMP
CN Ompanyt
CN OMZ
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS 3D CONCORD
DR 172964-80-6, 131959-78-9
MF C17 H19 N3 O3 S
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*,

L13 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2003 ACS

AB The effect of the H2 blockers cimetidine and ranitidine on drug-induced damage to gastric cell monolayers was evaluated in conditions independent of systemic factors and their **antiacid** properties. Monolayers of mucous cells from a human cell line MKN 28, obtained from human gastric adenocarcinoma, were studied. Cell damage was assessed qual. by trypan blue dye exclusion test and quant. by 51Cr release assay. Cimetidine and ranitidine protected cultured cells against damage induced by Na taurocholate decreasing taurocholate induced 51Cr release by 36 and 28%, resp. Cimetidine was protective in concns. lower than ranitidine. This protection was not prevented by the prostaglandin synthesis inhibitor indomethacin nor by the sulfhydryl (SH) blocker N-ethylmaleimide. Incubation with cimetidine and ranitidine did not increase the prodn. of PGE2 by cultured cells nor did it affect the cellular level of SH compds. Cimetidine and ranitidine did not afford protection against damage induced by indomethacin and ethanol. Cimetidine (10-4M) increased ethanol-induced damage significantly. In conclusion (1) cimetidine and ranitidine protect gastric cells against taurocholate-induced damage in vitro, independently of their **antiacid** effect; (2) this protection is not mediated by PGE2 or SH compds.; (3) cimetidine and ranitidine do not protect gastric cells against damage induced by indomethacin and ethanol.

IT 51481-61-9, Cimetidine **66357-35-5**, Ranitidine
RL: **BIOL (Biological study)**
(drug-induced ulcer inhibition by, mechanism of)

ontg. antacid agents for preventing
interactions of sucralfate and other drugs)

IT 50-54-4, Quinidine sulfate 53-86-1, Indomethacin 57-41-0, Phenytoin 58-55-9, Theophylline, biological studies 69-09-0, Chlorpromazine hydrochloride 94-20-2, Chlorpropamide 144-55-8, Sodium hydrogen carbonate, biological studies 471-34-1, Calcium carbonate, biological studies 546-93-0, Magnesium carbonate 549-18-8, Amitriptyline hydrochloride 614-39-1, Procainamide hydrochloride 1309-48-4, Magnesium oxide, biological studies 2610-86-8, Warfarin potassium 3166-62-9, Methylbenactyzium bromide 12304-65-3, Hydrotalcite 12511-31-8, Magnesium aluminate metasilicate 15676-16-1, Sulpiride 15687-27-1, Ibuprofen 20830-75-5, Digoxin 21645-51-2, Aluminum hydroxide, biological studies 22071-15-4, Ketoprofen 22204-53-1, Naproxen 28041-93-2, Aluminum calcium p-aminosalicylate 51481-61-9, Cimetidine 54182-58-0, Sucralfate 65277-42-1, Ketoconazole 66357-35-5, Ranitidine 70458-96-7, Norfloxacin 76824-35-6, Famotidine 76963-41-2, Nizatidine 78273-80-0, Roxatidine 81789-85-7, Indenolol hydrochloride 93107-08-5, Ciprofloxacin hydrochloride
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(pharmaceuticals contg. antacid agents for preventing

L12 ANSWER 15 OF 149 CAPLUS COPYRIGHT 2002 ACS

TI Sulfonate-containing strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**

AB Sulfonate-contg. strong acidic ion-exchange resins, e.g. sulfonated polystyrene-divinylbenzene copolymer, are claimed as **inhibitors** of **Helicobacter** pylori **adhesion** and are useful for treatment of gastritis, gastric ulcer, and duodenal ulcer in combination with gastric acid secretion **inhibitors**.

IT Stomach
(acid secretion **inhibitors**; sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT Intestine, disease
(duodenum, ulcer; sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT Stomach, disease
(gastritis; sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT **Adhesion**, biological
Antiulcer agents
Drug interactions
Helicobacter pylori
(sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT Ion exchangers
(sulfonated; sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT Stomach, disease
(ulcer; sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

IT 9042-14-2, Dextran sulfate 9064-57-7, .lambda.-Carrageenan 11114-20-8, .kappa.-Carrageenan 104469-08-1, Fractogel PGM 2000
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sulfonate-contg. strong acidic ion-exchange resins as **inhibitors** of **Helicobacter** pylori **adhesion**)

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2001031576	A2	20010206	JP 2000-145066	20000517

L12 ANSWER 16 OF 149 CAPLUS COPYRIGHT 2002 ACS
TI A high molecular mass constituent of cranberry juice **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus
AB Because previous studies have shown that a high mol. mass constituent of cranberry juice **inhibited adhesion** of Escherichia coli to epithelial cells and coaggregation of oral bacteria, we have examd. its effect on the **adhesion** of **Helicobacter** pylori to immobilized human mucus and to erythrocytes. We employed three strains of H. pylori all of which bound to the mucus and agglutinated human erythrocytes via a sialic acid-specific adhesin. The results showed that a high mol. mass constituent derived from cranberry juice **inhibits** the sialic acid-specific adhesion of H. pylori to human gastric mucus and to human erythrocytes.
IT Sialic acids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(-specific **adhesion**; cranberry juice high mol. mass constituent **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus and erythrocytes)
IT Cell **adhesion**
Erythrocyte
Helicobacter pylori
Mucus
Stomach
(cranberry juice high mol. mass constituent **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus and erythrocytes)
IT Fruit and vegetable juices
(cranberry; cranberry juice high mol. mass constituent **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus and erythrocytes)
IT Cranberry
(juice; cranberry juice high mol. mass constituent **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus and erythrocytes)
IT Adhesins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(sialic acid-specific; cranberry juice high mol. mass constituent **inhibits** **Helicobacter** pylori **adhesion** to human gastric mucus and erythrocytes)

L12 ANSWER 17 OF 149 CAPLUS COPYRIGHT 2002 ACS
AB **Helicobacter** pylori is a major etiol. agent in gastroduodenal disorders. The **adhesion** of H. pylori to gastric epithelial cells is the initial step of H. pylori infection. **Inhibition** of H. pylori adhesion is thus a therapeutic target in the prevention of H. pylori infection. We have reported that rebamipide and ecabet sodium, mucoprotective antiulcer agents, independently **inhibit** H. pylori adhesion. However, the antiadhesion activity of each antiulcer agent was incomplete. Expts. were performed to evaluate the combined effect of rebamipide and ecabet sodium on H. pylori adhesion to gastric epithelial cells. MKN-28 and MKN-45 cells, derived from human gastric carcinomas, were used as target cells. Twelve clin. isolates of H. pylori were used in this study. We evaluated the effects of rebamipide and ecabet sodium, individually and in combination, on H. pylori adhesion to target cells quant. using our previously established ELISA. Rebamipide and ecabet sodium each partially inhibited H. pylori adhesion. In contrast, adhesion was almost completely inhibited by pretreating target cells and H. pylori with the combination of rebamipide and ecabet sodium. Our studies suggest that the synergistic antiadhesion activity of rebamipide and ecabet sodium is greater than that of each antiulcer agent alone.

L12 ANSWER 18 OF 149 CAPLUS COPYRIGHT 2002 ACS

TI **Inhibiting** of growth and **adhesion** of **Helicobacter** pylori using egg yolk antibodies

AB **Helicobacter** pylori is known as a key pathogen for chronic gastric and duodenal ulcers. Egg yolk antibody, IgY produced from chicken immunized with H. pylori antigen was tested for the **inhibition** of growth and **adhesion** of H. pylori to gastric epithelial cell, AGS. The colony forming of H. pylori was repressed by 30% using 1 mg/mL of IgY while that of E. coli was only 7% with the same amt. of IgY, which showed the growth **inhibition** of H. pylori was mainly due to the specific interaction between IgY and H. pylori. The **inhibition** of H. pylori adhesion to AGS was a high as 90% using 0.5 mg/mL of antibody only. More than 80% of H. pylori attached to AGS could be detached treating with the same amt. of IgY for one and a half hr. However, this effect was severely dependent on the H. pylori strains tested. The strain used for immunization of chicken was very sensitive to the antibody treatment but changing the test strain generally showed a variation in adhesion inhibition between 15 and 80%. Further studies are necessary to employ the egg yolk antibodies for the treatment of H. pylori in vivo.

IT Proteins, specific or class

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(OMP (outer membrane protein); growth and **adhesion** to gastric epithelium by **Helicobacter** pylori is **inhibited** by IgY to)

IT Immunoglobulins

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(Y; growth and **adhesion** to gastric epithelium by **Helicobacter** pylori is **inhibited** by)

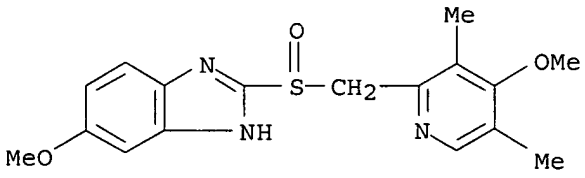
IT Stomach

(epithelium; growth and **adhesion** to gastric epithelium by **Helicobacter** pylori is **inhibited** by IgY)

BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CIN, CSCHEM, CSNB, DDFU, DIOGENES, DRUGNL, DRUGPAT, DRUGU, DRUGUPDATES, EMBASE, HSDB*, IPA, MEDLINE, MRCK*, PHAR, PHARMASEARCH, PIRA, PROMT, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU

(*File contains numerically searchable property data)

Other Sources: WHO



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PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2353 REFERENCES IN FILE CA (1957 TO DATE)
45 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2362 REFERENCES IN FILE CAPLUS (1957 TO DATE)

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